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Long-run trends of human aging and longevity

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Abstract Over the last 200 years, humans experienced a huge increase of life expectancy. These advances were largely driven by extrinsic improvements of their environment (for example, the available diet, disease prevalence, vaccination, and the state of hygiene and sanitation). In this paper, we ask whether future improvements of life expectancy will be bounded from above by human life span. Life span, in contrast to life expectancy, is conceptualized as a biological measure of longevity driven by the intrinsic rate of bodily deterioration. In order to pursue our question, we first present a modern theory of aging developed by bio-gerontologists and show that immutable life span would put an upper limit on life expectancy. We then show for a sample of developed countries that human life span thus defined was indeed constant until the mid-twentieth century but increased since then in sync with life expectancy. In other words, we find evidence for manufactured life span.

Keywords Human life span · Life expectancy · Aging · Compression of morbidity · Life-span extension

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1 Introduction

For economists, human life span is a given constant. It is the upper bound T that we put on top of the integral or sum sign when we compute expected lifetime utility of a representative agent. Life span differs conceptually from life expectancy. Although we frequently treat life- expectancy, a measure, which depends on the probability to survive from one period to the next, as a constant, this is an assumption made “for convenience”, since survival depends certainly on the macroeconomic environment (GDP per capita, doctors per square kilometer, etc.) and on individual economic decisions (nutrition, health expenditure, etc.).

While most of economic theory is based on the simplifying assumptions of either certain survival until a given end of life or a given age-unrelated survival probability per time unit of life (based on Yaari 1965 and Blanchard 1985), there exists also a, by now, rich literature that tries to endogenize survival and to incorporate empirically plausible survival probabilities into economic reasoning. To the best of our knowledge, however, there exists no research in economics on human life span.¹

With contrast to life expectancy, which is population specific and situation specific, life span is usually conceptualized as a *species-specific* characteristic (Arking 2006; Gavrilov and Gavrilova 1991). Life expectancy of a population of mice, for example, depends on the specific environment in the wild or in the laboratory. Life span of mice, in contrast, is independent from such conditions but it differs from the life span of fruit flies or elephants. Likewise, life expectancy of a particular human population differs across countries (England vs. Uganda) and over time (England today vs. 200 years ago). Life span of human beings, however, if it exists, should be invariant across populations. Probably all biologists agree to this notion of life span. But how to measure life span is—in contrast to life expectancy—less easily agreed upon.

Defining human life span as the maximum attainable age at death, as suggested in many general dictionaries and many older contributions in biology, is certainly misleading (Wilmoth 1999; Carey 2003). Conceptually, this idea is refuted by the insight that “however old we are, our probability to die within the next hour is never equal to one” (Jacquard 1982). Empirically, it has been refuted by the observation that maximum age at death has been continuously on the rise for at least 140 years (Wilmoth and Robine 2003).

The simple fact that the “sample size” of people who ever lived on earth is continuously rising lets us expect that the maximum ever-observed life length will rise

¹For economic theory with realistic survival probabilities, see, for example, Büttler (2001), Boucekkinne et al. (2002), French (2005), Bloom et al. (2007), Bullard and Feigenbaum (2007), Hansen and Imrohorglu (2008), Heijdra and Romp (2008, 2009), and Kalemli-Ozcan and Weil (2010). A few economic papers, among them Ben-Porath (1967), Cervellati and Sunde (2005), and Hazan (2009), could be interpreted as studies on the economic impact of increasing life span, although they rarely call it so and ignore its biological foundation.

as time proceeds. This is impressively shown by Finch and Pike (1996). They define life span T as the estimated age at death of the last survivor of a population such that $S(x) = 1/N$ is the probability to be the last man standing out of N . Plugging $1/N$ into an empirically estimated Gompertzian survival function, they obtain a life span of 105 years for $N = 10^3$ and 114 years for $N = 10^7$. In general, the dependence of maxima on sample size is a well-known fact of the statistics of extreme values (Gumbel 1958).

From these observations, it should be clear that life span, in contrast to life expectancy, cannot be defined as a mere statistical measure without biological foundation. We need some understanding about the biological mechanism of aging in order to infer an *intrinsic* mechanism that governs the pace at which our bodies deteriorate. Following standard reasoning in modern biology by taking aging and death as stochastic processes (Arking 2006), we are interested in whether a regularity exists which is common to all humans independently from environmental and genetic characteristics. From this regularity, if it exists, we try to infer life span not as an absolute maximum but as the characteristic length of life.²

We begin our pursuit of human life span in Section 2 by introducing two very strong empirical regularities, the Gompertz–Makeham law of mortality and the compensation effect of mortality. These empirical regularities are theoretically supported by reliability theory as shown by Gavrilov and Gavrilova (1991) and other bio-gerontologists. This led to the development of a novel theoretically founded conceptualization of human life span. We sketch the essence of this theory and its consequences in Section 2.3. The most important quantitative implication is that human life span can be inferred from the estimated compensation effect of mortality. In Section 3, we show that human life span if it was immutable would lead to a rectangularization of the survival curve and thus would put an upper bound on future advances of human longevity.

In Section 4, we estimate the parameters of the Gompertz–Makeham equation using the method of nonlinear least squares and data on age-specific mortality rates from the Human Mortality Database (2010). We then use the estimated parameters to analyze the compensation effect of mortality and infer human life span. We observe a time-invariant compensating effect until the mid-twentieth century for all countries in our sample. For the second half of the twentieth century, we find evidence for a secular increase of human life span. In other words, we find that human ingenuity has effectively interfered with nature and has created “manufactured lifetime” (Carnes and Olshansky 2007). Finally, we show that life expectancy in the second half of the last century increased in sync with life span. To a large and increasing extent, we are

²Notice that our analysis at the population level cannot be generalized to population subgroups. Olshansky SJ et al (2012), for example, document diverging trends in life expectancy by socioeconomic status and race for the USA. In 2008, college educated white males lived more than 14 years longer than black males with less than high school education. For women, this difference is still more than 10 years. Avendano et al. (2011) points out that highly educated Americans are relatively similar to Europeans in terms of life expectancy, while the gap is quite large at lower education levels. One possible explanation for these stylized facts is different access to universal health care in Europe and the USA. Strulik (2011) proposes an explanation of the rising education gradient in an economic life cycle model with endogenous educational choice.

living longer because technological progress has changed the way we age and the way our bodies decay.

Our study continues the work of Strehler and Mildvan (1960) and Gavrilov and Gavrilova (1991). Compared to these earlier works, we used a larger sample consisting of more countries and covering a much longer period of time. This allowed us not only to confirm the earlier result of a time-invariant compensating effect (time-invariant life span) until the mid-twentieth century but also, based on new data from the late twentieth century, to derive the novel result of a secular increase of human life span. This finding in turn allowed us to offer a novel explanation for the observation of “broken limits to life expectancy” (Oeppen and Vaupel 2002): since about the 1970s, later born humans can expect to live longer at least partly because human life span itself is on the rise.

2 Human life span: theory

2.1 The Gompertz–Makeham law of mortality

All theories of human life span are based on a very strong empirical regularity, the Gompertz–Makeham law of mortality. It originates from actuary Benjamin Gompertz (1825) who observed that there exists a long period of life, ranging from about 30 to 90 years of age, for which age and mortality are log-linearly related. Let $\mu(x)$ denote the *force of mortality*, that is the conditional probability to die at age x with a given survival of up to age x . The Gompertz law is then formally stated as $\mu(x) = R \exp(\alpha x)$. Noting that not all causes of death are age-related, Makeham (1860) added a constant, $A \geq 0$, which provides the famous Gompertz–Makeham formula:

$$\mu(x) = A + R e^{\alpha x}. \quad (1)$$

Taking both simplicity and precision into account, the Gompertz–Makeham formula is to the present day the most appropriate, concise, and widely used formal description of aging (Olshansky and Carnes 1997). Its parameters are estimated with great precision with R^2 above 0.9 not only for humans but also for species as different as yeast, fruit flies, and horses. The estimated coefficients, of course, differ greatly, reflecting the large variation in life span across species (Arking 2006).

Using the Gompertz–Makeham law and solving $\dot{S}(x)/S(x) = -\mu(x)$, we obtain the unconditional probability to survive to age x . Given that $S(0) = 1$, we arrive at Eq. 2:

$$S(x) = \exp \left(-Ax - \frac{R}{\alpha} (\exp(\alpha x) - 1) \right). \quad (2)$$

From $S(x)$, we can infer life expectancy (expected remaining years to live) at age x as $L(x) = \int_x^\infty S(a) da / S(x)$.

Over the last century, human life expectancy at birth increased by more than 20 years in most of the fully developed countries (Riley 2001). It is interesting to

investigate how these huge improvements of human longevity are captured by the Gompertz–Makeham law. For this purpose, it is helpful to isolate the Makeham parameter A because it reflects age-unrelated forces of mortality, i.e., background mortality (Bongaarts 2005) or extrinsic mortality (Carnes and Olshansky 2007). We expect prevention, eradication, or cure of age-unrelated diseases to be reflected in changes of A . In contrast, any progress with respect to the aging process itself would be reflected in a change of the age-dependent Gompertz parameters R and α . As can be seen from Eqs. 1 and 2, a lower R would mean that humans start out in better health (in youth) and a lower α would mean that the speed of the aging process is slowed down. However, as shown in Section 2.3, we expect that α and R do not change independently as long as human life span is immutable.

Over the last two centuries, background mortality went down dramatically in today's fully developed countries (for Sweden, for example, from $5.5 \cdot 10^{-3}$ to $4.8 \cdot 10^{-4}$; Gavrilov and Gavrilova 1991). With A being close to zero, future advances in life expectancy will have to come from improvements of age-dependent mortality. In other words, if technological progress could affect only background mortality but not the intrinsic rate of bodily decay, the observed trend of improving life expectancy at birth by about 3 months per year of birth (Oeppen and Vaupel 2002) would not be sustainable in the future. Inspired by this fact, some gerontologists have concluded that future life expectancy at birth will unlikely exceed 85 years (Fries 1980; Carnes and Olshansky 2007). The problem with such a hypothesis is that—although the virtually zero background mortality leaves no scope for improvement—we cannot (yet) see any slowdown or convergence of life expectancy in the data (Wilmoth 1997, 1999). This means that recent advances of life expectancy must have originated from a process that is reflected by a change of the Gompertz parameters.

2.2 The compensation effect of mortality

The Gompertz parameters α and R , which are estimated with high precision for a *given* population, differ actually across sexes, across countries, and over time. This means that, while all humans seem to age according to a common general law of mortality, the specific parameters governing this law depend on sex, provenance, and time. Furthermore, R and α seem to depend also on geography and tend to fall (in the case of R) or rise (in the case of α) with economic development. Because of their instability, the Gompertz parameters as such are thus not suitable to identify human life span.

The Gompertz parameters, however, are not changing independently from each other but, strikingly, in a specific way such that they preserve an inverse association between α and the log of R . This fact is known as the Strehler–Mildvan correlation or the compensation effect of mortality (Strehler and Mildvan 1960; Gavrilov and Gavrilova 1991). It is exemplarily shown for Sweden in Figs. 1 and 2. Figure 1 shows the long-run trends for α and R . Over time, the slope parameter α tends to rise and the level parameter R tends to fall. This means that, over time, Swedish women tend to age at a faster speed but start out at lower initial mortality. Below we show the generality of this phenomenon across countries and how it can be explained by increasing initial redundancy in the reliability theory of aging.

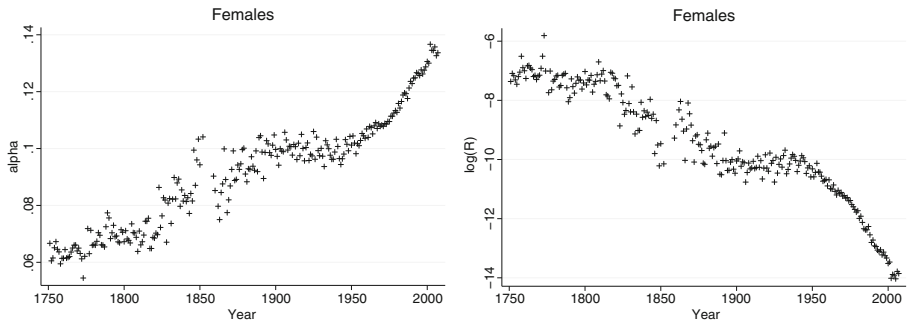


Fig. 1 Aging trends for Swedish females in 1751–2005. *Left panel:* Development of α over time. *Right panel:* Development of R over time

Formally, the compensation effect of mortality states that

$$\log(R_{it}) = \log(M) - \alpha_{it} \cdot T \quad (3)$$

in which R_{it} and α_{it} are the population- and year-specific parameters of the Gompertz law and M and T are invariant parameters of the compensation effect of mortality. Figure 2 shows the correlation for Swedish females and males.

To see how the compensation effect inspired a general theory of human aging, insert Eq. 4 into country- and sex-specific age-dependent mortality $\mu_i^x \equiv \mu(x) - A = R_i \exp(\alpha_i x)$ to obtain

$$\mu^x = M e^{\alpha(x-T)}. \quad (4)$$

Observe that age-dependent mortality exhibits a fixed point. Equation 4 predicts that all men (and likewise all women) of a country's population share a common force of mortality M at age T independently from the year or century of birth. In other words, any improvement in the initial force of mortality μ^0 is compensated by a faster increase of μ^x with age. The final step in the derivation of human life span is to conclude that T is approximately constant across countries. This has been found by Gavrilov and Gavrilova (1991) for 209 human life tables. They thus interpret the

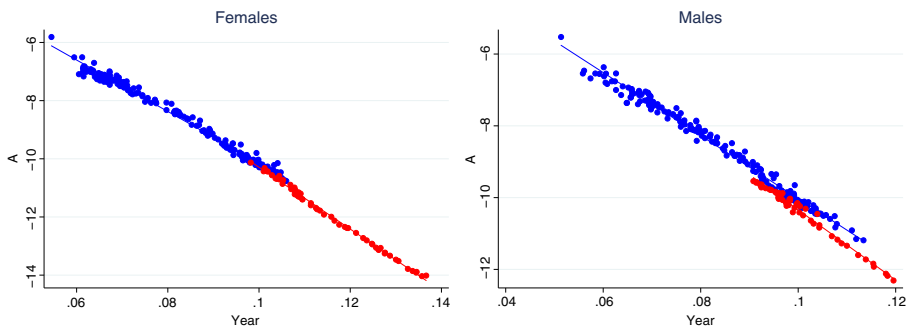


Fig. 2 The compensation effect of mortality in Sweden in 1751–2005. Correlation between $\log(R)$ and α for males (*right*) and females (*left*). Observations before 1950 are marked *blue*, and observations after 1950 are marked *red*

focal point (T, M) as a species-specific constant. For humans, the point estimate was $T = 95$ years, identified by Gavrilov and Gavrilova as “the life span of human beings.”

The difference between the Strehler–Mildvan correlation and the compensation effect is that Strehler and Mildvan (1960), in their original approach, inferred the correlation from the Gompertz law, without the Makeham amendment. In order to infer life span properly, however, the Gompertz parameters have to be derived from the Gompertz–Makeham law, with explicit consideration of background mortality A . Otherwise, results would be biased and changes in the extrinsic rate of mortality would be absorbed by the parameters governing the intrinsic rate of mortality. This has been demonstrated by Gavrilov and Gavrilova (1991) who called the unbiased correlation the “compensation effect of mortality.” Controlling for A is of particular importance when background mortality is high, that is in the analysis of historical data and of contemporaneous less developed countries.

2.3 Reliability theory

The strong regularities identified by Gompertz law and the compensation effect suggest that all humans share a common mechanism of aging, a common process according to which individual bodies lose function over time and bodily failures and impairments are accumulated. The pursuit to rationalize these phenomena has inspired an application of reliability theory, originally developed to study mechanical failures in engineering (Barlow and Proschan 1975), and produced the modern theory of aging (Arking 2006). Its main idea is based on redundancy within the human body which is depleted over time. This notion of aging as accelerated loss of organ reserve is in line with the mainstream view in the medical science. For example, initially, as a young adult, the functional capacity of human organs is estimated to be tenfold higher than needed for survival (Fries 1980). It accords also well with the modern view in biology, which conceptualizes aging and death as driven by accidental stochastic shocks on the molecular level (Arking 2006). Here, we sketch a basic theory by Gavrilov and Gavrilova (1991, 2001) and refer to their work as well as to Novoseltsev (2006), Finkelstein (2008), and Milne (2008) for extensions.

A common characteristic of all reliability-based models is that organisms are conceptualized as complex systems consisting of essential parts (e.g., organs, tissue) connected in series, which are in turn built of smaller entities connected in parallel. Parallel connectivity means that reliability theory is built upon the idea of redundancy. Another common theme is a stochastic failure rate for the basic entities. The notion of aging as driven by a stochastic process helps to explain the “unfair” nature of human fate, i.e., why we actually observe large differences of aging at the individual level. Reliability theory can explain why individuals raised under equal conditions and/or built from the same genes (monozygotic twins) can age and eventually die in very different ways. At the same time, the model provides a toehold to explain how population- (e.g., country-) specific characteristics and the environment early in life have a bearing on aggregate aging behavior of entire populations and/or subpopulations.

Suppose an organism consists of m irreplaceable blocks, i.e., blocks connected in series such that the organism dies if one block fails. Each block consists of n elements, connected in parallel with age-independent failure rate λ . The probability for a block to expire before age x is given by $F(x) = [1 - \exp(-\lambda x)]^n$. Suppose that many elements are initially defect. The probability of an initially functioning element is given by q . It can then be shown that the failure rate of the organism (i.e., the mortality rate) is given by Eq. 5:

$$\mu(x) \approx Re^{\alpha \cdot x} \quad \text{where} \quad \begin{cases} R & \equiv mc\lambda k \exp(-k) \\ \alpha & \equiv k\lambda, \end{cases} \quad (5)$$

in which $k \equiv nq$ is the mean number of initially functioning elements and c is a constant to provide a unitless result. The model thus explains Gompertz' law. Taking log's of R , we get $\log(R) = \log(cmk\lambda) - k$, and inserting $\alpha = k\lambda$, we arrive at $\log(R) = \log(M) - \alpha T$, with $M \equiv \alpha mc$ and $T \equiv 1/\lambda$. The model thus explains the compensation effect of mortality.

For an interpretation of the result, note that T is uniquely pinned down by λ , the age-independent failure rate of an element. If λ is a species-specific constant, then the model supports a unique focal point T , i.e., a species-specific life span. Across species, T depends inversely on the robustness of its nonaging elements. The compensation effect, according to this simple model of reliability, then means that any variation number of initially functioning elements, $k = nq$, is compensated by a proportional reduction of the number blocks m such that M stays constant.

Suppose that available nutrition (for mother and child) and disease exposure early in life have shaped k . Taking the historic improvement in nutrition and health into account, the model then predicts that, with ongoing economic development, people start out much better at young age but are aging faster. Consequently, survival prospects and life expectancy have improved at any age up to age T .

In other words, a time-invariant compensation effect of mortality would imply that better health care and nutrition or, more generally, improvements of the economic environment through technological progress have increased life expectancy "only" through improving somatic redundancy (for example, more healthy body cells; Fogel 1994; Fogel and Costa 1997). In order to improve life span T , technological progress needs to have a bearing on λ , that is on the intrinsic rate of bodily decay.

3 Implications of time-invariant human life span

3.1 Compression of morbidity

The notion of a constant life span T is sometimes expressed as "rectangularization" or as "compression of morbidity" (Fries 1980). These concepts are illustrated in Fig. 3. The left panel shows an improvement of the age-dependent force of aging according to the Strehler–Mildvan correlation. The slope parameter α increases from

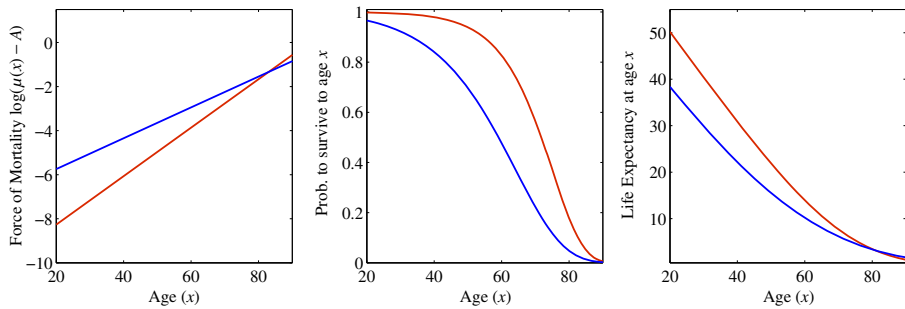


Fig. 3 Aging under compensation of mortality. $A = 0.002$, $\alpha = 0.08$ (blue lines) and $\alpha = 0.11$ (red lines), $M = \exp(-0.07)$, $R = M \exp(-\alpha \cdot T)$, $T = 83$

0.08 (blue line) to 0.11 (red line). This is about the actually observed average increase for women during 20th century in our sample of countries. The level parameter R is assumed to adjust in a way that supports a fixed point at $T = 83$.

The middle panel shows the implied unconditional survival rate $S(x)$ and conveys the idea of rectangularization. Compared to the beginning of the century, a higher share of women reach an old age of, say, 70 years and expire then more quickly during their last years before death. Visually, the survival curve becomes closer to rectangular over time. This implies that gains in life expectancy, for example, brought forward by technological progress in medicine, decrease with age as well, as demonstrated in the panel on the right-hand side of Fig. 3. Improvements in health at earlier stages of life lead to a faster deterioration in old age, a compression of morbidity, such that women reaching an age of T share the same force of mortality at the beginning and at the end of the century.

For comparison, Fig. 4 shows the effect on survival and life expectancy caused by an improving level parameter R without compensation effect. The slope parameter α is held constant. In this case, the curves shift to the right at all ages, indicating that aging as such has been postponed at all ages and providing a visible increase of life expectancy also for the old (see also Vaupel 2010). In terms of theory, the inherent

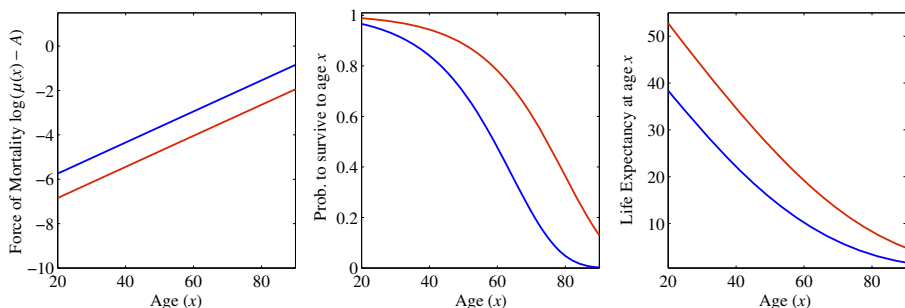


Fig. 4 Manufactured life span. $A = 0.002$, $\alpha = 0.011$, $R = 2.7 \cdot 10^{-5}$ (blue lines), and $R = 0.9 \cdot 10^{-5}$ (red lines)

failure rate of the elements of which humans are constructed λ has been manipulated. Human life span T increased.

3.2 The future of longevity

In this section, we show that if the compensation effect of mortality is time invariant and M and T are species-specific constants, then future gains of life expectancy are bounded from above. This means that the notion of broken limits to life expectancy (Oeppen and Vaupel 2002) inferred from extrapolation of past trends would be ill-considered. Actually, there would exist a certain limit to life expectancy. In order to show this, we insert the compensation effect of mortality, $R = M \exp(-\alpha T)$, into Eq. 1. Assuming that A is zero (which is a reasonable approximation for contemporaneous fully developed countries), we get the force of mortality $\mu = M \exp[\alpha(x - T)]$. The implied survival at age x is $S(x) = \exp(y) \exp[-M/\alpha \exp(\alpha(x - T))]$ with $y \equiv M/\alpha \exp(-\alpha T)$. For analytical simplicity, we focus on life expectancy at birth, which is given by Eq. 6:

$$L(0) = \exp(y) \int_0^\infty \exp[-M/\alpha \exp(\alpha(x - T))] dx. \quad (6)$$

Substituting $u = M/\alpha \exp[\alpha(x - T)]$ into Eq. 6 provides (Eq. 7):

$$L(0) = \exp(y) \frac{1}{\alpha} \int_y^\infty \exp(-u) \frac{1}{u} du = \exp(y) \frac{1}{\alpha} \Gamma(0, y) \quad (7)$$

in which Γ is the incomplete gamma function. Applying the mean value theorem of integral calculus, we know that there exists a ξ with $y < \xi < \infty$ such that

$$\int_y^\infty \exp(-u) \frac{1}{u} du = \frac{1}{\xi} \int_y^\infty \exp(-u) du = \frac{1}{\xi} \exp(-y).$$

Thus, $L(0) = 1/(\alpha\xi)$, implying, since $y < \xi < \infty$, that $L(0) < 1/(\alpha y)$. Taking the derivative of life expectancy with respect to life span, we obtain

$$\frac{\partial L(0)}{\partial T} = \left[L(0) + \frac{\exp(y)}{\alpha} \frac{\partial \Gamma}{\partial y} \right] \frac{\partial y}{\partial T} = \left[L(0) - \frac{1}{\alpha y} \right] \cdot (-\alpha y) > 0 \quad (8)$$

because $L(0) < 1/(\alpha y)$. That is, life expectancy and life span are positively correlated. Analogously, we obtain $\partial L(0)/\partial M < 0$.

If the compensation effect of mortality is time invariant and M and T are constants, the only way to improve life expectancy is through α . In order to obtain the effect of α on $L(0)$, we compute

$$\frac{\partial L(0)}{\partial \alpha} = -\frac{L(0)}{\alpha} + \left[L(0) - \frac{1}{\alpha y} \right] \frac{\partial y}{\partial \alpha} = -\frac{L(0)}{\alpha} - \left(\frac{1}{\alpha} + T \right) y L + \frac{1}{\alpha} \left(\frac{1}{\alpha} + T \right).$$

The sign of the derivative is ambiguous and there exists an extremum for $\alpha = \alpha^*$ where $\partial L / \partial \alpha = 0$, that is at life expectancy,

$$L(0, \alpha^*) = \frac{\frac{1}{\alpha^*} + T}{1 + y + \alpha^* y T}. \quad (9)$$

After some algebra, the second derivative is obtained as

$$\frac{\partial^2 L}{\partial \alpha^2} \Big|_{\alpha^*} = \frac{1}{\alpha^3} \cdot \frac{1}{1 + y + \alpha^* y T} \cdot \left[y \alpha^* T (1 + \alpha^* T)^2 - 1 \right].$$

From this, we conclude that the extremum is a maximum if

$$(1 + \alpha T)^2 \leq \frac{1}{\alpha y T} = \frac{1}{M T} e^{\alpha T} \Rightarrow e M T \cdot (1 + \alpha T)^2 < e^{1 + \alpha T}.$$

Since the slope of the exponential function is generally steeper than the slope of the quadratic, we conclude that, for sufficiently large α , there exists a maximum life expectancy. This in turn means that life expectancy is effectively bounded from above by life span. Since y converges very rapidly to zero with rising α , we expect maximum life expectancy to be about $T + 1/\alpha^*$.

Figure 5 visualizes these results for life span $T = 85$. As the force of mortality α increases, the logarithm of initial mortality $\log(R)$ declines according to the compensation effect (Eq. 3). This is shown by the dashed green line in Fig. 5, assuming that $M = 0.26$. Accordingly, life expectancy at birth increases steeply for small α and reaches an almost indiscernible maximum of 85.78 years at 1.27. Afterwards, life expectancy converges towards T from above (blue solid line). The panel on the right-hand side demonstrates robustness of this result by—hypothetically—considering alternative survival rates M at age T . Noting that a perpetually increasing α under the compensation effect of mortality implies a rectangularization of the survival curve

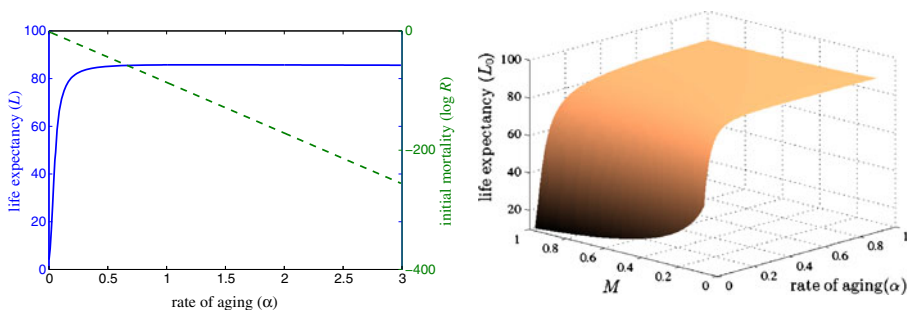


Fig. 5 Life expectancy is bounded by life span. *Left panel:* Life expectancy (blue solid line) and initial mortality $\log(R)$ under the compensation effect of mortality (Eq. 3) (green dashed line) for alternative α and $M = 0.26$. The—almost indiscernible—maximum life span is assumed for $\alpha = 1.27$. *Right panel:* Life expectancy for alternative values of α and M . *Both panels:* Lifespan $T = 85$

and that $\lim_{\alpha \rightarrow \infty} L(0) = T$ provides another definition of life span: it is the maximum life expectancy of a species under the compensation effect of mortality. This implies that the observation of broken limits to life expectancy (Oeppen and Vaupel 2002) is actually inconsistent with a time-invariant compensation effect. In order to allow for “unlimited” life expectancy, life span of humans must be modifiable.

4 Empirical evidence

In this section, we use the established methodology and investigate whether human life span is constant or modifiable. For that purpose, we use data on 1×1 period death rates from the Human Mortality Database (2010). 1×1 means that the data come for single years of age in 1-year time intervals. The Human Mortality Database contains detailed population and mortality data for 37 countries (there are multiple data series for France, Germany, New Zealand, and the UK). The period of data coverage differs from country to country. For Sweden, for example, data are available way back to 1751, while for Chile, data coverage starts only in 1992. We confine our analysis to countries that have full data coverage from 1900 through 1999 (12 countries) or 1950 through 1999 (26 countries). This way, we ensure a consistent sample and that our results are not due to changes in the sample composition.

4.1 The Gompertz parameters

We begin with estimating the parameters of the Gompertz–Makeham equation (Eq. 1) by gender for all countries and years with the method of nonlinear least squares. It is well known from the literature that the Gompertz–Makeham equation does not hold for young and very old people. We thus restrict the sample to ages 30–90. Both Gompertz parameters are estimated with very high precision. The R^2 is around 0.99 in all regressions. Figure A.1 in the [Electronic supplementary material](#) shows the distribution of the estimated parameters divided by their respective standard errors. There is not a single case of an insignificant α parameter. In the 1950–1999 sample, we lose one country-year observation due to an insignificant estimate of the R parameter for Finland. In the 1900–1999 sample, we lose 14 country-year observations due to insignificant estimates of the R parameter for Finland and New Zealand.

The parameter estimates for α and $\log(R)$ are reported in Tables 1 and 2. For better visualization, we break the data down into 25-year periods and report period averages. The overall trend shown in Section 2 for Sweden is clearly visible across all countries: α increases and R decreases over time. There are also interesting gender differences: α is higher for females than for males, whereas R is higher for males than for females. To simplify the exposition, we have computed in the bottom line of Tables 1 and 2 the average α and R for the respective time periods and gender. On average, α increased for females from 0.089 in 1900–1924 to 0.112 in 1975–1999. For males, the increase is more modest, starting from 0.088 in 1900–1924 and reaching 0.091 in 1975–1999. For females, R decreased by more than one order of magnitude from $\exp(-9.14) = 1.07 \cdot 10^{-4}$ in 1900–1924 to $\exp(-11.70) = 8.3 \cdot 10^{-6}$ in 1975–1999. Again, the decrease is somewhat more modest for males.

Table 1 Slope parameter α across countries and time

Country	1900–1924		1925–1949		1950–1974		1975–1999	
	Male	Female	Male	Female	Male	Female	Male	Female
Australia					0.081	0.097	0.092	0.111
Austria					0.087	0.102	0.095	0.118
Belgium	0.086	0.090	0.090	0.093	0.089	0.103	0.091	0.116
Bulgaria					0.080	0.088	0.088	0.102
Canada					0.085	0.099	0.089	0.109
Czech Republic					0.087	0.101	0.086	0.107
Denmark	0.098	0.093	0.095	0.095	0.094	0.105	0.088	0.111
England and Wales	0.078	0.081	0.086	0.092	0.085	0.100	0.089	0.106
Finland	0.090	0.094	0.076	0.090	0.084	0.100	0.087	0.114
France	0.089	0.088	0.088	0.096	0.089	0.107	0.099	0.125
Germany					0.089	0.103	0.093	0.114
Hungary					0.092	0.100	0.084	0.103
Ireland					0.089	0.095	0.088	0.103
Italy	0.085	0.079	0.095	0.095	0.093	0.103	0.094	0.119
Japan					0.088	0.100	0.101	0.124
Netherlands	0.089	0.088	0.094	0.094	0.095	0.105	0.090	0.118
New Zealand	0.079	0.084	0.090	0.092	0.085	0.101	0.089	0.108
Northern Ireland					0.085	0.093	0.086	0.103
Norway	0.100	0.100	0.100	0.102	0.096	0.107	0.094	0.119
Portugal					0.089	0.099	0.093	0.111
Scotland	0.079	0.081	0.086	0.090	0.084	0.095	0.084	0.099
Slovakia					0.094	0.103	0.084	0.107
Spain					0.088	0.099	0.097	0.120
Sweden	0.101	0.100	0.099	0.099	0.097	0.106	0.099	0.120
Switzerland	0.084	0.083	0.087	0.092	0.093	0.106	0.100	0.127
USA					0.079	0.098	0.087	0.105
Average	0.088	0.089	0.090	0.094	0.088	0.101	0.091	0.112

Summarizing, across all countries over the last century, the trend shows that people become more healthy in young age but are aging faster as they get older.³

³In a related study, Bongaarts (2005) estimates the parameters of the Gompertz law for 14 countries during the period 1950–2000 and finds a “nearly constant” slope parameter (α in our notation) and a secularly falling level parameter (R in our notation). It is tempting to conclude from Bongaarts’ finding of decreasing R that human life span has risen over the last half of the twentieth century. On a closer look, however, this evidence is insufficient for a conclusion on human life span. Firstly, α and R are not species-specific parameters; they vary across countries and sexes. Secondly, it is misleading to compare α and R at the same (absolute) scale. Inspection of Eq. 1 shows that a unit change of α contributes as much to the force of mortality as a unit change of the logarithm of R .

Table 2 Level parameter $\log(R)$ across countries and time

Country	1900–1924		1925–1949		1950–1974		1975–1999	
	Male	Female	Male	Female	Male	Female	Male	Female
Australia					−8.55	−10.22	−9.73	−11.71
Austria					−9.00	−10.47	−9.86	−12.09
Belgium	−8.75	−9.18	−9.08	−9.56	−9.19	−10.52	−9.52	−12.00
Bulgaria					−8.62	−9.36	−9.15	−10.51
Canada					−9.05	−10.50	−9.57	−11.66
Czech Republic					−8.90	−10.26	−8.85	−10.96
Denmark	−9.90	−9.59	−9.67	−9.74	−9.74	−10.81	−9.34	−11.64
England and Wales	−8.15	−8.56	−8.78	−9.51	−8.83	−10.37	−9.38	−11.20
Finland	−9.05	−9.48	−8.06	−9.23	−8.70	−10.20	−9.17	−11.80
France	−8.93	−9.03	−8.92	−9.74	−9.22	−10.95	−10.30	−12.97
Germany					−9.13	−10.54	−9.63	−11.77
Hungary					−9.33	−10.20	−8.78	−10.66
Ireland					−9.19	−9.88	−9.20	−10.81
Italy	−8.65	−8.18	−9.52	−9.66	−9.58	−10.58	−9.89	−12.29
Japan					−9.02	−10.28	−10.55	−12.85
Netherlands	−9.06	−9.10	−9.60	−9.69	−9.83	−10.87	−9.52	−12.30
New Zealand	−8.43	−9.02	−9.29	−9.68	−8.92	−10.53	−9.46	−11.43
Northern Ireland					−8.85	−9.80	−9.11	−10.86
Norway	−10.21	−10.35	−10.27	−10.52	−9.95	−11.05	−9.83	−12.37
Portugal					−9.13	−10.23	−9.67	−11.52
Scotland	−8.19	−8.54	−8.73	−9.33	−8.68	−9.92	−8.86	−10.49
Slovakia					−9.61	−10.45	−8.82	−10.95
Spain					−9.18	−10.28	−10.15	−12.47
Sweden	−10.22	−10.17	−10.03	−10.16	−9.96	−10.86	−10.26	−12.45
Switzerland	−8.50	−8.52	−8.87	−9.40	−9.53	−10.83	−10.41	−13.08
USA					−8.55	−10.35	−9.35	−11.29
Average	−9.00	−9.14	−9.24	−9.68	−9.16	−10.40	−9.55	−11.70

4.2 Survival probability

We next use the average parameters of the Gompertz equation to compute the unconditional probability to survive to age x :

$$S(x) = \exp\left(-\frac{R}{\alpha}(\exp(\alpha x) - 1)\right). \quad (10)$$

Figure 6 shows the survival functions for average females and males in 1900–1924 (solid lines) and 1975–1999 (dashed lines). Two salient phenomena can be observed.

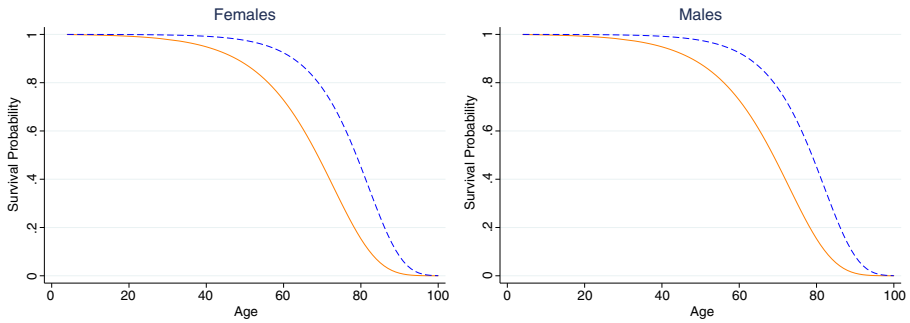


Fig. 6 Survival function in 1900–1924 and 1975–1999. *Dashed blue line:* Survival function in 1975–1999. *Solid orange line:* Survival function in 1900–1924

First, a rectangularization or compression of morbidity is clearly visible. In 1900–1924, the decline of the survival probability starts already around age 20 whereas in 1975–1999, survival probability stays close to unity until around age 40 and then declines more rapidly than in the first quarter of the century. Second, in addition to the rectangularization of the survival function, we observe also that the survival function shifts to the right from period 1900–1924 to 1975–1999. The right shift is not uniform across all ages, but clearly very old people are benefitting from improved survival probabilities as well. As explained in Section 2, this finding is inconsistent with a time-invariant compensation effect of mortality. It is a first indication that human life span might be modifiable.

In Fig. A.2 in the [Electronic supplementary material](#), we redo the analysis country by country. We take the period averages of α and R for the first available period and compare the obtained survival function to the survival function in 1975–1999 for each country. It turns out that the pattern that we have illustrated for averages—rectangularization cum right shift of the survival function—is universal across all countries.⁴

4.3 Human life span

The observations from the previous section suggest that life span T according to the definition from the compensation effect of mortality is not constant but changing over time. We substantiate this finding by estimating the compensation effect. In the model,

$$\log(R_{it}) = \beta_0 + \beta_1 \cdot \alpha_{it} + \epsilon_{it}. \quad (11)$$

β_1 represents the negative life span $-T$ and β_0 represents $\log(M)$, the mortality rate shared by all humans of age T . Exploratory data analysis in Fig. A.3 in the [Electronic supplementary material](#) suggests that the slope coefficient T indeed changes

⁴Yashin et al. (2001) arrive at a similar conclusion using period and cohort data for a smaller sample of countries (France, Japan, Sweden, and the USA).

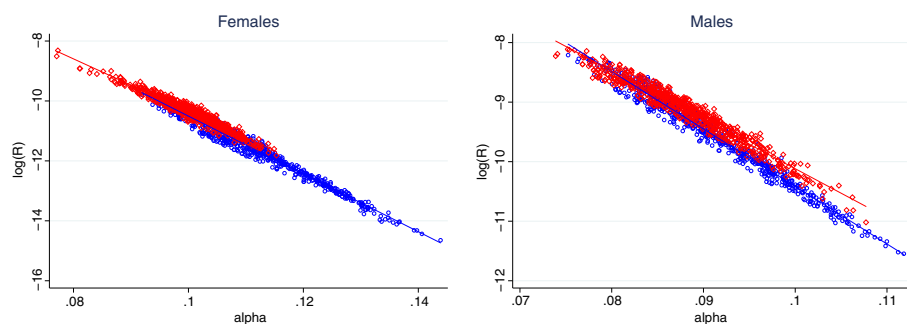


Fig. 7 Strehler–Mildvan correlation in 1950–1999. Data points 1975–1999 (*blue circles*) and 1950–1974 (*red diamonds*). The *solid lines* represent the pooled OLS fit for each period. The countries in the sample are Australia, Austria, Belgium, Bulgaria, Canada, Czech Republic, Denmark, England and Wales, Finland, France, Hungary, Iceland, Ireland, Italy, Japan, Netherlands, New Zealand, Northern Ireland, Norway, Portugal, Scotland, Slovakia, Spain, Sweden, Switzerland, and the USA

over time. Pooling the observations over a too long period would lead to misleading results. We thus focus on the 26 countries, for which we have a full data from 1950 to 1999 and estimate Eq. 11 with pooled OLS for 1950–1974 and 1975–1999, respectively. The result is visualized in Fig. 7. Red circles identify data points in the 1950–1974 period and blue circles reflect the 1975–1999 period. It is clearly visible that, in the later period, an improvement of $\log(R)$ was associated with a somewhat smaller increase of α than in the earlier period, implying that the slope—representing human life span—increased in absolute value during the twentieth century.

Table 3 reports the estimation results. During the second half of the twentieth century female life span increased from 88 years to about 96 years and male life span increased from 82 years to almost 97 years. These numbers should be taken with some caution. Pooling the data over (arbitrarily chosen) 25-year periods clearly has some unwanted side effects if the slope coefficient changes over time. The main

Table 3 Human life span in 1950–1999

	1950–1974		1975–1999	
	Male	Female	Male	Female
T	82.2 (0.73)	87.6 (0.86)	96.6 (0.62)	96.4 (0.67)
$\log(M)$	−1.90 (0.06)	−1.59 (0.09)	−0.76 (0.06)	−0.86 (0.08)
R^2	0.95	0.94	0.97	0.97

Standard errors in parentheses. The countries in the sample are Australia, Austria, Belgium, Bulgaria, Canada, Czech Republic, Denmark, England and Wales, Finland, France, Germany, Hungary, Ireland, Italy, Japan, Netherlands, New Zealand, Northern Ireland, Norway, Portugal, Scotland, Slovakia, Spain, Sweden, Switzerland, and the USA

Table 4 Human life span in 1900–1999

	1900–1924		1925–1949		1950–1974		1975–1999	
	Male	Female	Male	Female	Male	Female	Male	Female
T	86.9 (0.79)	89.0 (0.96)	86.5 (0.93)	86.4 (1.28)	89.7 (0.96)	95.2 (1.21)	94.7 (0.55)	93.5 (0.58)
$\log(M)$	−1.313 (0.07)	−1.27 (0.09)	−1.42 (0.08)	−1.54 (0.12)	−1.24 (0.09)	−0.81 (0.12)	−0.95 (0.05)	−1.23 (0.07)
R^2	0.98	0.97	0.97	0.94	0.97	0.95	0.99	0.99

Standard errors in parentheses. The countries in the sample are Belgium, Denmark, England and Wales, Finland, France, Italy, Netherlands, New Zealand, Norway, Scotland, Sweden, and Switzerland

point that we want to make here is that the slope coefficient, that is human life span, was not time invariant.⁵

Expanding life span, however, is a fairly recent phenomenon. Extending the sample period over the whole last century provides supporting evidence for the notion of invariant life span during the first half of the century. As shown in Table 4, we estimate a life span of about 87 years for men and 89 years for women in the 1900–1924 period. Then, in the 1950–1974 period, female life span took off and male life span followed in the 1975–1999 period. We interpret these results as follows. During the first half of the twentieth century (and presumably also earlier in human history), the data support the notion of an invariant human life span of about 87–89 years. Observable improvements of life expectancy during that period originated from declining background mortality (sanitation, vaccination) and from a reduction of R in association of movement along the mortality compensation line, indicating better (initial) physiological conditions (better nutrition, e.g., Fogel and Costa 1997). Then, in the later twentieth century, “something” happened. According to reliability theory, these events effectively manipulated the failure rate, that is the intrinsic rate of bodily decay.

4.4 Life span and life expectancy

Finally, we would like to explore the link between life span and life expectancy empirically. For that purpose, we estimate Eq. 11 annually from 1950 to 1999 for the sample of 26 countries for which we have consistent data and compare it with the

⁵Recently, Zheng et al. (2011) arrived at a similar result. Their estimates, however, were unfortunately based on the Gompertz equation, without the Makeham amendment. Since their sample contained many less developed countries, the bias incurred from ignoring background mortality was potentially large (see Gavrilov and Gavrilova 1991). This potential bias may explain why they arrived at a much higher estimate of life span (referred to as the age of zero vitality according to the Strehler–Mildvan methodology) for a sample containing many developing countries than we do for a sample of fully developed countries.

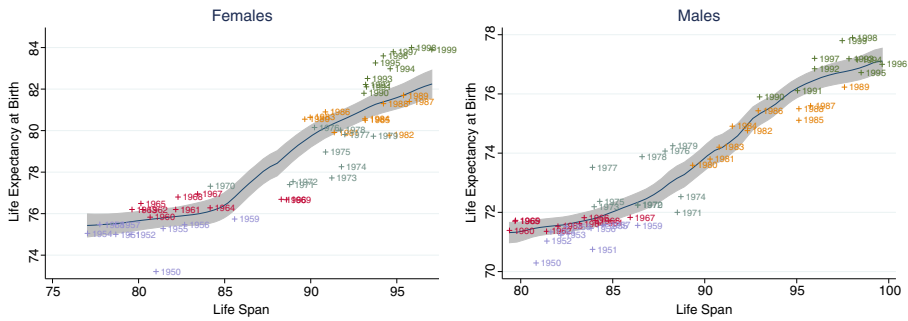


Fig. 8 Life span and “frontier” life expectancy. *Abscissa*: Life span T_l obtained from a simple linear cross-country regression of $\log(R)$ on α (Eq. 11). *Ordinate*: Frontier life expectancy taken from Oeppen and Vaupel (2002). The *solid line* is a fitted spline with a 95 % confidence interval around it (shaded in gray). Countries as for Table 3

“frontier life expectancy” calculated by Oeppen and Vaupel (2002).⁶ We visualize the combined data in Fig. 8 in a simple scatter plot and add a spline fit with a 95 % confidence interval to it.

Interestingly, life span and frontier life expectancy are virtually uncorrelated for the first 20 years of the observation period, which further corroborates the idea of improving life expectancy through lower background mortality or through better health and nutrition in line with an invariant compensation effect. Then, from about the 1970s and onwards, we observe a strong positive correlation between the two variables, indicating that recent improvements in life expectancy were indeed driven at least partly by expanding human life span.

The notion of an expanding human life span is helpful to rationalize why other researchers have not (yet) observed any convergence of increasing life expectancy (Wilmoth 1997; Vaupel 2010). Predictions of a certain limit to life expectancy—for example, that it should not exceed 35 years at age of 50 (Olshansky et al. 1990)—are presumably made under the wrong perception of an invariant human life span, in which case they would have been indeed fully justified and understandable as the analysis in Section 3.2 has shown. Unlike other species, however, humans seem to be able to modify life span such that there is no limit of life expectancy visible in the data.

5 Conclusions

In this paper, we have introduced to the economic science a theoretical foundation of human life span based on two strong empirical regularities, the Gompertz–Makeham law and the compensation effect of mortality. We have estimated the parameters of

⁶Frontier life expectancy is defined, according to Oeppen and Vaupel, as the life expectancy of the country, which has the highest life expectancy in the world for a given year.

the Gompertz–Makeham model and found a remarkable long-run trend. Contemporaneous humans start out as young adults much more healthy than their forefathers a century ago but they are also aging faster. For a long time of our history, this trend was consistent with the observation of rising life expectancy under an invariant life span.

If life span was indeed immutable, as it presumably is for other animals, future improvements in life expectancy would soon end. Life expectancy, as we have shown in this paper, is bounded from above by life span. For the second half of the twentieth century, however, we were able to present evidence for expanding life span. It increased by about 8 years. For the last quarter of the twentieth century, we find furthermore that life expectancy increased in sync with life expectancy, a phenomenon that explains why other researchers have observed broken limits to life expectancy (Oeppen and Vaupel 2002).

It is beyond the scope of the present paper to identify the cause of expanding life span. Yet theory suggests that the potential candidates have to be fairly recent events which manipulated the failure rate of human body parts. The most natural candidates are probably regenerative medicine and replacement surgery. To be more concrete, heart bypass operation and dialysis machines are two prominent examples of medical innovations of the second half of the twentieth century which prolonged human lives by replacing the functional performance of failed organs.

The notion of modifiable life span is important for economists, policymakers, as well as theorists. Taking the capital T as constant may lead to severe misjudgments about the future of population aging and the financial stability, meaningfulness, and efficiency of social security and public health systems. Also, on the individual level, the awareness of modifiable and potentially further rising life span beyond the “natural” improvements of life expectancy may have quite dramatic effects on life-cycle decisions like schooling, savings, and retirement. Reconsidering the conventional life-cycle theory and integrating the notion of expanding life span is a challenging task for future research.

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References

- Arking R (2006) The biology of aging: observations and principles. Oxford University Press, Oxford
- Avendano M, Kok R, Glymour M, Berkman L, Kawachi I, Kunst A, Mackenbach J (2011) Do Americans have higher mortality than Europeans at all levels of the education distribution?: a comparison of the

- United States and 14 European countries. In: Crimmins EM, Preston SH, Cohen B (eds) *International differences in mortality at older ages*. National Academies Press, Washington, DC
- Barlow RE, Proschan F (1975) *Statistical theory of reliability and life testing: probability models*. Holt, Rinehart and Winston, New York
- Ben-Porath Y (1967) The production of human capital and the life cycle of earnings. *J Polit Econ* 75:352–365
- Blanchard OJ (1985) Debt, deficits, and finite horizons. *J Polit Econ* 93:223–247
- Bloom DE, Canning D, Moore M (2007) A theory of retirement. NBER Working Papers 13630
- Bongaarts J (2005) Long-range trends in adult mortality: models and projection methods. *Demography* 42:23–49
- Boucekkine R, De la Croix D, Licandro O (2002) Vintage human capital, demographic trends, and endogenous growth. *J Econ Theory* 104:340–375
- Bullard J, Feigenbaum J (2007) A leisurely reading of the life-cycle consumption data. *J Monet Econ* 54:2305–2320
- Büttler M (2001) Neoclassical life-cycle consumption: a textbook example. *Econ Theory* 17:209–221
- Carey JR (2003) Life span: a conceptual overview. *Popul Dev Rev* 29:1–18
- Carnes BA, Olshansky SJ (2007) A realist view of aging, mortality, and future longevity. *Popul Dev Rev* 33:367–381
- Cervellati M, Sunde U (2005) Human capital formation, life expectancy, and the process of development. *Am Econ Rev* 95:1653–1672
- Finkelstein MS (2008) *Failure rate modeling for risk and reliability*. Springer, London
- Finch CE, Pike MC (1996) Maximum life span predictions from the Gompertz mortality model. *J Gerontol* 51:183–194
- Fogel RW (1994) Economic growth, population theory, and physiology: the bearing of long-term processes on the making of economic policy. *Am Econ Rev* 84:369–395
- Fogel RW, Costa D (1997) A theory of technophysio evolution, with some implications for forecasting population, health care costs, and pension costs. *Demography* 34:49–66
- French E (2005) The effects of health, wealth, and wages on labour supply and retirement behaviour. *Rev Econ Stud* 72:395–427
- Fries JF (1980) Aging, natural death, and the compression of morbidity. *New Engl J Med* 303:130–135
- Gavrilov LA, Gavrilova NS (1991) *The biology of human life span: a quantitative approach*. Harwood Academic Publishers, London
- Gavrilov LA, Gavrilova NS (2001) The reliability theory of aging and longevity. *J Theor Biol* 213:527–545
- Gompertz B (1825) On the nature of the function expressive of the law of human mortality, and on a new mode of determining the value of life contingencies. *Philos Trans R Soc Lond* 115:513–583
- Gumbel EJ (1958) *Statistics of extremes*. Columbia University Press, New York
- Hansen GD, Imrohoroglu S (2008) Consumption over the life cycle: the role of annuities. *Rev Econ Dyn* 11:566–583
- Hazan M (2009) Longevity and lifetime labor supply: evidence and implications. *Econometrica* 77:1829–1863
- Heijdra BJ, Romp WE (2008) A life-cycle overlapping-generations model of the small open economy. *Oxf Econ Pap* 60:88–21
- Heijdra BJ, Romp WE (2009) Retirement, pensions, and ageing. *J Pub Econ* 93:586–604
- Human Mortality Database (2010) University of California, Berkeley (USA), and Max Planck Institute for Demographic Research (Germany). Available at www.mortality.org (data downloaded on 08/25/2010)
- Jacquard A (1982) Heritability of human longevity. In: Preston HS (ed) *Biological and social aspects of mortality and the length of life*. Ordina Editions, Liege
- Kalemli-Ozcan S, Weil DN (2010) Mortality change, the uncertainty effect, and retirement. *J Econ Growth* 15:1–27
- Makeham WM (1860) On the law of mortality and the construction of annuity tables. *J Inst Actuar* 8:301–310
- Milne EMG (2008) The natural distribution of survival. *J Theor Biol* 255:223–236
- Novoseltsev VN (2006) Mathematical modeling in biology: systems capable to live and die. *Autom Remote Control* 67:835–855
- Oeppen J, Vaupel JW (2002) Broken limits to life expectancy. *Science* 296:1029–1031
- Olshansky SJ, Carnes BA (1997) Ever since Gompertz. *Demography* 34:1–15

- Olshansky SJ, Carnes BA, Cassel C (1990) In search of Methuselah: estimating the upper limits to human longevity. *Science* 250:634–640
- Olshansky SJ et al (2012) Differences in life expectancy due to race and educational differences are widening, and many may not catch up. *Health Aff* 18:1803–1813
- Riley JC (2001) *Rising life expectancy: a global history*. Cambridge University Press, Cambridge
- Strehler BL, Mildvan AS (1960) General theory of mortality and aging. *Science* 132:14–21
- Strulik H (2011) Health and education: understanding the gradient. University of Hannover, Discussion Paper 487
- Vaupel JW (2010) Biodemography of human aging. *Nature* 464:536–542
- Wilmoth JR (1997) In search of limits. In: Wachter KW, Finch CE (eds) *Between Zeus and the Salmon: the biodemography of aging*. National Academy of Sciences, Washington, DC, pp 38–63
- Wilmoth JR (1999) Rectangularization revisited: variability of age at death within human populations. *Demography* 36:475–495
- Wilmoth JR, Robine JM (2003) The world trend in maximum life span. *Popul Dev Rev* 29:239–257
- Yaari ME (1965) Uncertain lifetime, life insurance and the theory of the consumer. *Rev Econ Stud* 32:137–1350
- Yashin AI, Begun AS, Boiko SI, Ukraintseva SV, Oeppen J (2001) The new trends in survival improvement require a revision of traditional gerontological concepts. *Exp Gerontol* 37:157–167
- Zheng H, Yang Y, Land KC (2011) Heterogeneity in the Strehler–Mildvan general theory of mortality and aging. *Demography* 48:267–290